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Neural Correlates of Performance Monitoring in Daily and Intermittent Smokers

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Abstract

OBJECTIVES—Despite efforts that have increased smoking regulation, cigarette taxation, and social stigma, cigarette smoking remains the leading cause of preventable death worldwide, and a significant personal and public economic burden. In the U.S., intermittent smokers comprise approximately 20% of all smokers and represent a stable, non-dependent group that may possess protective factors that prevent the transition to dependence. One possibility is that intermittent smokers have intact CNS frontal regulatory and control mechanisms that enable resistance to nicotine-induced changes.

METHODS—The present study measured inhibitory control using a flanker task and a go-nogo continuous performance tasks in daily dependent smokers, intermittent non-dependent smokers, and nonsmokers. Event-related potential (ERP) measures of were concurrently recorded to measure performance monitoring via event-related negativity (ERN) and error positivity (Pe) components during error trials for each task.

RESULTS—In both tasks, behavioral and ERN measures did not differ between groups; however, amplitude of the Pe component was largest among intermittent smokers.

CONCLUSIONS—Thus, intermittent smokers differed from both daily smokers and nonsmokers on error processing, potentially revealing neuroprotective cognitive processes in nicotine dependence.

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SIGNIFICANCE—A better understanding of factors that mediate behavioral regulation may provide novel treatment approaches that help individuals achieve controlled smoking or cessation.

Keywords

Performance Monitoring; Error-Related Negativity; Event-Related Potentials; Smoking; Substance Dependence

INTRODUCTION

Difficulty regulating behavior and monitoring performance outcomes plays a significant role in the development and maintenance of addiction (Chiu et al., 2008; de Wit, 2008, Sokhadze et al., 2008). Drug dependent individuals have difficulty inhibiting the response to drugs or drug cues, especially during withdrawal (Li and Sinha, 2008). A compromised self-regulatory system in dependent smokers may drive relapse rates to be as high as 50% in the first week and 95% in the first year following cessation, despite a desire to quit by nearly 70% of individuals (CDC, 2011a; Hughes, 2007). Smokers report reduced self-control, increased impulsiveness, and inability to resist temptation, all of which may contribute to relapse (Coggins et al., 2009). Impairments on laboratory tasks of inhibitory control and executive function are evident in abstinent states, even following three months of cessation, compared to satiated states, which may contribute to failed cessation efforts (Billieux et al., 2010; Dawkins et al., 2009; Harrison et al., 2009). Compared to nonsmokers, imaging studies show neuroadaptation of frontostriatal brain regions involved in regulating inhibitory control, as evidenced by reduced frontal grey matter volumes and densities, increases in neural response to drug cues, and reduced anterior cingulate metabolic activity in chronic smokers during abstinence (Feil et al., 2010). The ability to regulate cognitive processes and adjust behavior for optimal performance requires online monitoring of actions and subsequent outcomes (Ridderinkhof et al., 2004). In smokers, reduced neural response following error commission suggests potential difficulties in performance monitoring that may potentiate relapse or continued smoking despite negative health consequences (Franken et al., 2010; Luijten et al., 2011b).

A subset of smokers report feeling in control of their smoking behavior, an absence of withdrawal symptoms, and higher cessation rates (Coggins, 2009; Shiffman, 1989; Shiffman et al., 1990). These self-reported “some day” smokers, also referred to as “chippers” or intermittent smokers (ITS), comprise 22% (9.9 million) of the U.S. smoking population and this number is believed to be increasing (CDC, 2011b; Shiffman et al., 2012). Their ability to avoid dependence cannot be attributed to differences in smoking topography (e.g., number of puffs, puff duration, inter-puff/cigarette interval) or changes in blood-nicotine concentration (Brauer et al., 1996; Coggins et al., 2009; Shiffman, 1989). Compared to daily, dependent smokers, long-term ITS report having an internal locus of control and greater self-control compared to regular smokers (Coggins, 2009; Kassel et al., 1994; Shiffman and Paty, 2006). Surprisingly, the neurocognitive factors that allow ITS to avoid dependence have received remarkably little attention.

Behavioral measures of inhibitory control have not been investigated previously in ITS. The Eriksen Flanker Task targets selective inhibition and conflict control, where participants must respond quickly to identify the middle letter (or arrow) in trials when it is flanked by congruent or incongruent letters (or arrows). The Go/No-go continuous performance task targets sustained attention and behavioral control where participants must inhibit a prepotent motor response to a frequently occurring “Go” stimulus when an infrequent `No-go' stimulus is presented. These tasks reflect the ability to control behavior prior to motor initiation, such as inhibiting smoking-related cues, or the automaticity of reaching for a cigarette when it is available, respectively, and may be especially relevant in the planning and single instances of drug seeking or use. Overall, studies of inhibitory control in smokers have produced mixed results, finding that smokers had impaired accuracy in some (Luijten et al., 2011a: Go/No-go task) but not all studies (Dinn et al., 2004): Go/No-go task, Stroop Color-Word task; Franken, 2010: Flanker task; Luijten, 2011b: modified Flanker task). Impaired performance on measures sensitive to behavioral control, such as the Stop Signal task (Bilieux, 2010) and the Go/No-go and Anti-saccade tasks (Spinella, 2002) has been found to correlate with increased smoking rate and dependence.

The medial frontal cortex and associated regions are involved in monitoring unfavorable outcomes, response errors and conflict, and decision uncertainty (Ridderinkhof et al., 2004). Event-related potential (ERP) recordings during performance of the inhibitory control tasks provide an evaluation of online performance monitoring and behavioral modification when errors are detected. The Error-Related Negativity (ERN) and Error Positivity (Pe) ERP components are believed to index performance monitoring in tasks that induce cognitive and response conflicts. The ERN is a negative-voltage potential with a fronto-central scalp distribution that occurs approximately 50–100ms following the commission of an error (Gehring et al., 1993). The Pe is a positive-voltage potential with a fronto-central or centro-parietal scalp distribution that occurs approximately 200–400 ms following an erroneous response (Arbel and Donchin, 2009; Overbeek, 2005). The ERN is associated with automatic error detention or conflict monitoring, whereas the Pe is associated with the awareness and motivational significance of an error (Arbel and Donchin, 2009; Falkenstein et al., 2000; Gehring et al., 2012; Nieuwenhuis et al., 2001; Overbeek et al., 2005; Pontifex et al., 2010; Shalgi et al., 2009; Yeung et al., 2004). Generation of the ERN has been localized to the dorsal anterior cingulate cortex (ACC) with potential contributions from the pre-supplementary motor area and the lateral prefrontal cortex, whereas the less-understood Pe may be comprised of a fronto-central waveform originating from the medial frontal cortex and a late centro-parietal waveform generated by the superior parietal cortex and rostral ACC (Arbel & Donchin, 2009; Gehring et al., 2012; Herrmann et al., 2004; Overbeek et al., 2005; Pontifex et al., 2010). Recent studies support that the Pe co-varies with the stimulus-locked P300 ERP, associated with attentional salience and novelty detection (Polich, 2007; Ridderinkhof, Ramautar, & Wijnen, 2009; Shalgi et al., 2009).

Ascending dopaminergic projections, which densely innervate the medial frontal cortex, release the primary neurotransmitter responding to errors in reward prediction. Phasic dopamine changes may play a role in adjusting behavior to improve task performance via reinforcement learning principles (Overbeek et al., 2005). The ERN has been consistently found to be sensitive to dopamine neurotransmission, however the role of dopamine has

been less certain for the P3—and Pe response (Gehring et al., 2012; Overbeek et al., 2005). There has been some support of dopaminergic mediation of frontal P300 (P3a), but studies have found no influence of dopamine on the Pe response (Gehring et al., 2012; Overbeek et al., 2005; Polich and Criado, 2006). In substance dependence, frontostriatal dysregulation can impair the selection and maintenance of task/goal-relevant information while suppressing inappropriate responses or representations (Dawkins et al., 2009; Feil et al., 2010). Specifically, substance dependence is associated with impairments in the dorsolateral prefrontal cortex (involved in attention, goal identification, and selection) and the ACC (involved in assessment of consequences and error detection) (Feil et al., 2010; Goldstein & Volkow, 2002), which may contribute to deficits in inhibitory control and performance monitoring.

Imaging studies support that substance abuse populations have shown decreased error-related activity in the anterior cingulate cortex, a region believed to be involved in ERN and Pe generation (Gehring, 2012; Olvet and Hajcak, 2008). Studies of drug users have found disrupted ERN response in regular users cocaine (Franken et al., 2007), and alcohol (Schellekens et al., 2010) and disrupted Pe/P300 in users cannabis (Fridberg et al., 2013), cocaine (Franken et al., 2007), and alcohol (Polich & Ochoa, 2004; Rodriguez Holguin et al., 1999). Fridberg et al. (2013) found no ERN differences between controls and chronic cannabis users. In participants at risk for alcoholism, smoking was found to moderate the P300 such that smoking accounted for more variance of the decreased P300 than alcohol risk (Polich & Ochoa, 2004). Studies specific to smoking have found reduced amplitudes of the ERN (Luijten et al., 2011b) and Pe (Franken, 2010; Luijten, 2011b) in smokers compared to nonsmokers during inhibitory control tasks (Franken, 2010; Luijten, 2011b). However, Franken et al. (2010) found no ERN differences between controls cigarette smokers. In an oddball task, increased P3a amplitude in response to the distractor shows that the P300 is sensitive to acute nicotine administration both in low-use and high-use chronic smokers, suggesting nicotine-dependent alterations in the brain mechanisms contributing to P300 generation (Polich and Criado, 2006). Finally, acute abstinence from smoking resulted in reduced ERN amplitude during a flanker task (Schlienz et al., 2013). Insensitivity to error commission and suboptimal error detection and processing may contribute to the continuation of maladaptive behaviors despite negative consequences.

Understanding the factors contributing to resistance against nicotine dependence, specifically the ability to control behavior, is crucial for resolving why certain individuals have the ability to control their smoking behavior and for developing more successful prevention and intervention strategies for those who are unable to quit smoking. The purpose of this study is to evaluate whether ITS demonstrate better inhibitory control than dependent smokers as measured by behavioral and ERP measures. We expected that smokers would commit more commission errors (false positives) and exhibit faster reaction times (RTs) than ITS and nonsmokers during inhibitory control tasks, reflecting greater behavioral disinhibition. We expected that ITS would fall between smokers and nonsmokers in terms of those measures. Similarly, amplitudes of performance monitoring ERPs (ERN and Pe) were expected to be greatest in nonsmokers, intermediate in ITS, and smallest in dependent smokers. Overall, reduced inhibitory control and error processing deficits were predicted to be associated with increased dependence.

MATERIALS AND METHODS

Participants

Thirty nonsmokers, 31 intermittent smokers (ITS), and 22 daily smokers (DS) were recruited from the local community and paid $10 per hour for participation. Participants were excluded for a history of electroconvulsive therapy, neurological illness, or drug dependence based on DSM-IV criteria. Participants were also excluded for current anxiety disorders or major depression, use of psychotropic medications, or current drug abuse. Males drinking more than 14 drinks per week and females drinking more than 7 drinks per week were also excluded. Participants who reported using marijuana more than once per week were excluded. Minimum age for participation was 20 years old to allow for sufficient time for progression to nicotine dependence (Brook et al., 2010; Dierker et al., 2008). Maximum participation age was 50 years old. All participants received detailed information about the study protocol and gave written and oral informed consent. The local institutional review board approved the study.

Groups were classified according to the following criteria. Nonsmokers smoked <10 cigarettes in their lifetime and had not smoked in the past month. ITS 1) in the preceding 90 days, smoked on ≥10 days or smoked ≥20 cigarettes; 2) smoked <27 days per month for the preceding six months; 3) were not attempting to quit; 4) smoked in any capacity for the previous three years; and 5) scored <4 on the Fagerström Test for Nicotine Dependence (FTND) (Baker et al., 2007; Heatherton et al., 1991). DS 1) smoked <25 cigarettes per day, daily for ≥12 months; and 2) scored ≥4 on the FTND. Demographic information for the three groups is presented in Table 1.

Procedure

Interviews—Potential participants were screened by phone to determine their eligibility for the study. Study eligibility was confirmed in the laboratory using the demographics and screening module of the Structured Clinical Interview for Axis-I disorders (SCID-I; First et al., 1997), with follow-up questions from additional modules when necessary.

Smoking Behavior and CO levels—Questionnaires were mailed to individuals who qualified for the study and were completed prior to or after the lab session. Nicotine dependence was assessed using the Fagerström Test for Nicotine Dependence (FTND; Heatherton, 1991) and the Nicotine Dependence Syndrome Scale (NDSS; Shiffman et al., 2004). Nicotine dependence was defined as a score ≥4 on the FTND (Baker, 2007). Current nicotine withdrawal was assessed before and after the three-hour testing session using a revised Wisconsin Smoking Withdrawal Scale (WSWS; Hendricks et al., 2006). Breath carbon monoxide (CO) was measured before and after the testing session (piCO+, Bedfont Scientific Ltd., Rochester, UK). A criterion of six to eight CO ppm differentiates smokers from nonsmokers (Pearce and Hayes, 2005). Smoking measures are presented in Table 1.

Inhibitory Control Tasks—The Eriksen Flanker Task is designed to assess selective inhibition of distracting information and attentional control (Eriksen and Eriksen, 1974). In the version used in the present study, participants identified the middle letter of five letters

presented on the computer screen. Participants were told that their goal was to respond quickly and accurately to each trial. Figure 1 is a schematic of the events occurring on each trial. Following a block of 20 practice trials, participants completed four blocks of 100 trials, with a rest period provided after every block. Congruent and incongruent trials were presented at an equal rate and trials were randomized within each block. The computer recorded accuracy and RTs for each trial. The Go/No-go Continuous Performance Task is designed to measure sustained attention and behavioral response inhibition (Fridberg et al., 2013). In this Go/No-go version, participants made a two-choice discrimination between "X" and "O" stimuli that were rapidly presented on the computer screen. Stimuli were counterbalanced for presentation rates of rare (20%) or frequent (80%). Participants were instructed to respond quickly and accurately to each trial. Figure 1 is a schematic of the events occurring on each trial. Participants completed 30 practice trials where the stimuli were presented at an equal rate, followed by four blocks of 125 experimental trials. Trials were randomized within each block, and rest periods were provided after every block. The computer recorded accuracy and RTs for each trial.

Electrophysiological Assessment—The electroencephalogram was sampled continuously (1000 Hz sampling rate, 0.1–200 Hz bandpass filter) from 34 Ag/AgCl electrodes mounted in a cap (EasyCap, GmbH) and referenced to the nose (Torpey et al., 2012; Wiswede et al., 2009; Zirnheld et al., 2004). Two Ag/AgCl electrodes placed above and below the participant's left eye recorded bipolar vertical electrooculogram (vEOG). Neuroscan SynAmps I digitized the EEG, and electrode impedances were maintained at <10 kOhm. Participants completed the EEG recording session while comfortably seated in a sound-attenuated room. The Flanker and Go/No-go task order was randomized across participants. Stimuli were presented on a Power Macintosh computer using SuperLab stimulus presentation software. Stimuli were presented on a 17-inch CRT monitor centered 70 cm away from the participants.

For offline processing, the EEG was segmented into 1000 ms epochs, including a 200 ms pre-response baseline using Brain Vision Analyzer software (Brain Products, GmbH). The data were baseline corrected and filtered using a 0.1–40 Hz (24 dB/octave roll-off) using a Butterworth filter. Epochs were corrected for ocular artifacts using the Gratton et al. (1983) algorithm. Epochs with voltage exceeding ±100 μV at any site were automatically excluded from further analyses. Participants with fewer than ten accepted error trials were excluded from analysis (Olvet and Hajcak, 2009). Insufficient trials after artifact rejection resulted in the exclusion of four nonsmokers, five ITS, and one DS from Flanker analysis and four nonsmokers, two ITS, and one DS from Go/No-go analysis. The number of segments accepted for signal processing did not differ significantly between groups for Flanker task or Go/No-go task (Table 2).

Data Analysis: One-way Analysis of Variance (ANOVA) with between-subjects factor of group (3) was run for every self-report dependent variable. Although nonsmokers were more educated than DS (Table 1), years of education was not a significant covariate for any dependent measure when included in analysis of covariance (ANCOVA), and was therefore not included in the analysis. For the Flanker and Go/No-go behavioral data, group error rates

and post-error slowing were compared using ANOVA. Post-error slowing was calculated as the mean RT for post-error correct trials minus the mean RT for pre-error correct trials (Dutilh et al., 2012). In addition, accuracy and RTs were compared using a 3 (Group) × 2 (Stimulus Type: Congruent or Incongruent for Flanker, Frequent or Rare for Go/No-go) repeated-measures ANOVA. Follow-up ANOVAs and post-hoc Fisher's Least Significant Difference (LSD) test were used to resolve significant main effects or interactions for post-hoc analysis. Greenhouse-Geisser corrections for nonsphericity were applied when appropriate. One ITS did not complete the Flanker task and the Go/No-go task due to computer malfunction. Data from two nonsmokers were excluded from analysis due to commission of only one error and performance at chance levels.

For EEG data analysis, response-locked ERP averages were constructed using error trials for each participant, and did not include trials when the participant did not respond. The ERN and Pe components were measured at midline electrode sites (Fz, FCz, Cz, CPz, and Pz) based on previous research (Overbeek, 2005; Torpey, 2012). The ERN was automatically detected as the maximally negative voltage between 50 before to 100 ms after the incorrect response, and the Pe was automatically detected as the maximum positive voltage between 100 and 250 ms after the incorrect response. Peak amplitude was measured as the mean of the maximal voltage and its surrounding 20 data points (i.e., maximal value ±10 data points). Because the ERN was maximal at FCz and the Pe was maximal at Cz for both tasks across the entire participant sample, group comparisons focused on latency and amplitude at these sites. ANOVA was used to determine group differences and Greenhouse-Geisser corrections were applied when appropriate. Effect size estimates for analysis of variance were determined with partial $\eta 2$ (partial $\eta 2 = .01$ is a small effect size, .06 is a medium effect size, and .14 is large effect size) (Kittler et al, 2007). Data from one nonsmoker were excluded from analysis in the Flanker task for having an ERN voltage value differ from other participants by more than four standard deviations.

RESULTS

Behavioral Outcomes

Eriksen Flanker Task—For the assessment of accurate RTs, a group (3) × condition (2: congruent, incongruent) repeated measures ANOVA found faster RTs in the congruent condition ($F[1,77]=271.83$, $p<.001$) (Table 3). There was no main effect of group ($F[2,77]=.90$) and no group × condition interaction ($F[2,77]=.81$). For the assessment of error commission, a group (3) × condition (2) repeated measures ANOVA found more errors in the incongruent condition ($F[1,77]=149.02$, $p<.001$). There was no main effect of group ($F[2,77]=.27$) and no group × condition interaction ($F(2,77)=.976$). Finally, no group differences were found for post-error slowing ($F[2,77]=1.01$, $p=.368$).

Go/No-go Continuous Performance Task—For the assessment of accurate RTs on the Go/No-go task, a group (3) × condition (2: frequent, rare) repeated measures ANOVA found faster RTs in the frequent condition ($F[1,79]=323.98$, $p<.001$) (Table 3). There was no main effect of group ($F[2,79]=1.00$) and no group × condition interaction ($F[2,79]=.51$). For the assessment of errors, a group (3) × condition (2) repeated measures ANOVA found more

errors in the rare condition (F[1,79]=231.66, p<.001). There was no main effect of group (F[2,79]=.08) and no group × condition interaction (F[2,79]=.51). Finally, no group differences were found for post-error slowing.

Electrophysiological Measures

ERP outcomes are presented in Table 2. Figures 2–4 illustrate the grand averaged waveforms recorded during error trials of the Flanker and Go/No-go tasks. Figure 5 illustrates ERN and Pe component magnitudes calculated from the waveforms.

Eriksen Flanker Task—ANOVA revealed that ERN amplitude did not differ among groups. ERN latency was earlier for DS than ITS (p=.007). A trend for significant group effect for Pe amplitude (p=.052), with ITS having the highest response. No Pe latency differences were found.

Go/No-go Continuous Performance Task—ANOVA revealed that ERN amplitude and latency did not differ among groups. A main effect of group for PE amplitude revealed a greater amplitude for ITS compared to daily smokers (p=.017) and showed a trend for greater amplitude than nonsmokers (p=.051). No Pe latency differences were found.

DISCUSSION

The present study measured behavioral and electrophysiological indices of inhibitory control and performance monitoring in daily smokers, intermittent smokers (ITS), and nonsmokers. Daily smokers endorsed greater smoking rates and motivation, nicotine dependence, and risk factors for smoking than ITS. Groups did not differ in performance on the Flanker and Go/No-go task. Although we predicted that ERP amplitudes would decrease in magnitude as a mirror of smoking dependence, from none (high amplitude) to dependent (low amplitude), we found an absence of group differences for the ERN indicating that nicotine dependence alone is not responsible for deficits in executive control mechanisms. Furthermore, the amplitude of the error positivity (Pe) event-related potential was greatest among ITS during both inhibitory control tasks, indicating neural differences unique to ITS. Finally, faster ERN latency in DS may reflect acute nicotine effects.

The increased Pe component in ITS compared to daily smokers is intriguing in light of the absence of behavioral differences on the cognitive measures. The functional significance of the Pe is unclear and has not been well established in the context of drug abuse and dependence. Pe has been associated with conscious error processing and the motivational salience of an event (Falkenstein, 2000; Overbeek, 2005). Based on these interpretations of the Pe, ITS may have an increased sensitivity to error commission compared with both DS and nonsmokers. The increased response following error commission may indicate subsequent activation of executive functioning, reflecting heightened performance monitoring or greater sensitivity in circuitry that is also used to regulate smoking behavior. The Pe in nonsmokers may represent typical, or compared to ITS under-practiced performance monitoring, whereas monitoring in DS may be supplemented by the acute nicotine state.

A study of chronic cannabis users also found increased Pe ERPs and faster RTs for both correct and incorrect trials on the Go/No-go task relative to cannabis-naïve participants (Fridberg et al., 2013). The neural and reaction time differences were interpreted as a compensatory response following error commission. In the present study, a lack of behavioral differences among groups makes it difficult to provide evidence for a similar mechanism. However, it is important to note that cannabis use may reflect intermittent or non-dependent drug use behavior. Estimates of cannabis use in the U.S. population report high lifetime (42%) and past year (12%) prevalence, however the prevalence of abuse and dependence in ever users is low (9%) compared to other illicit drugs (e.g., nicotine conditional dependence: 40%) (Anthony et al., 1994; SAMHSA, 2010). Only 30% of chronic users in the Fridberg et al. (2013) study met DSM-IV criteria for dependence. Successful performance monitoring and regulatory behaviors may be associated with controlled drug use behavior and adaptive neural activation during error trials. Future studies are needed to examine individual differences in the Pe ERPs in individuals with chronic, non-dependent drug use.

The present results are consistent with those of previous studies that have found small or no differences between smokers and nonsmokers on inhibitory control tasks. In a Go/No-go task using picture stimuli, Luitjen et al. (2011a) found a trend for decreased accuracy on No-go trials in smokers compared to nonsmokers and no differences for RTs. Luitjen et al. (2011b) found no accuracy differences in a Flanker task that included pictures, though nonsmokers had increased post-error slowing following an error response, suggesting better performance monitoring. Franken et al. (2010) also found no accuracy or response time differences in a standard Flanker task. Smoking status, rate, and dependence correlated with poor performance on several behavioral inhibition tasks, including Go/No-go, anti-saccades, and delayed alteration task (Billieux, 2010; Spinella, 2002). It is possible that increased task difficulty and smoking rate/dependence may increase group differences due to recruitment of additional resources and greater engagement of control mechanisms.

ERP measures of performance monitoring have seldom been assessed in substance abuse/dependence populations, especially in smokers. Similar to the present study, Franken et al. (2010) found no ERN differences between smokers and nonsmokers on an Eriksen Flanker task. Luitjen et al. (2011b) suggest that the absence of ERN differences in the Franken et al. (2010) study was due to a lack of challenge and included smoking images during the Flanker task. The error rate in the current study was higher (11%) than the Luitjen et al. (2011b) rate (≈6.2%) for incongruent trials, and error rates did not differ across groups for either study. Although the smoking picture condition did not increase error rates compared to the neutral picture condition, Luitjen et al. (2011b) found ERN reductions for smokers compared to nonsmokers. Increasing task difficulty by requiring divided attention may have reduced available cognitive resources in smokers, resulting in reduced ERN compared to nonsmokers. Additionally, both Franken et al. (2010) and Luitjen et al. (2011b) found a reduced Pe for smokers compared to nonsmokers. Although their participants were similar to those in the present study in terms of nicotine dependence, absence of psychopathology, and other drug dependence, they were younger and they used both hands to respond, which may have required greater response control. An absence of ERN or Pe differences between

dependent smokers and nonsmokers in the present study was further supported by the fact that no differences were found in two separate tasks, the Flanker and Go/No-go.

Limitations and Future Directions

The comparison of satiated DS to ITS and nonsmokers in the present study was intended to avoid comparing a withdrawal state in DS to groups who do not experience withdrawal (ITS) or do not smoke. As a result, acute nicotine effects in DS may explain the lack of behavioral or electrophysiological differences when compared to nonsmokers. However, acute effects were not present in ITS who had increased Pe response that may be independent of nicotine state. Future studies are needed to test DS and ITS in peak and trough nicotinic states to resolve nicotine effects on ERPs in dependent and non-dependent individuals. A recent study found that response inhibition training in heavy drinkers resulted in reduced weekly alcohol intake (Houben et al., 2011). Therefore, the ability to monitor and exert control over behavior may be instrumental in achieving more-controlled smoking behavior or remission. Future studies are needed to investigate the ability to train ITS and DS in performance monitoring across different smoking and emotional/stress states as well as evaluate performance monitoring in long-term non-dependent substance to determine if the ERN or Pe are useful endophenotypes in substance dependence and its treatment.

There are additional limitations to the present study that should be considered. The participants' young age (*Median*=23) limits generalizability to older smokers with longer durations of smoking, who may demonstrate more pronounced neurobehavioral effects. Furthermore, only two daily smokers smoked more than a pack per day ($n \approx 25$ cigarettes), suggesting that this sample may be comprised primarily of relatively light to moderate smokers. In order to investigate differences due to nicotine alone, participation criteria excluded smokers with comorbid psychiatric disorders, which commonly occur in smokers. A larger sample size and greater variability in smoking history, comorbid diagnoses, gender, and ethnicity would the address these limitations.

Conclusions

A larger Pe response in ITS may be suggestive of higher responsiveness to error and may be associated with better performance monitoring. Differences in error processing could be used as risk factors in nicotine dependence, applied to evaluations of smoking cessation efficacy, and provide a deeper understanding of addiction processes in the brain.

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HIGHLIGHTS

- This is the first study to measure inhibitory control and event-related potentials (ERP) measures of performance monitoring in nondaily, non-dependent smokers.
- Monitoring and regulation of behavior is important for adaptive and optimal performance that is impaired in persons with substance dependence.
- Increased Error Positivity ERP response in nondaily smokers reflects cognitive processes that may prevent the transition to dependent smoking.

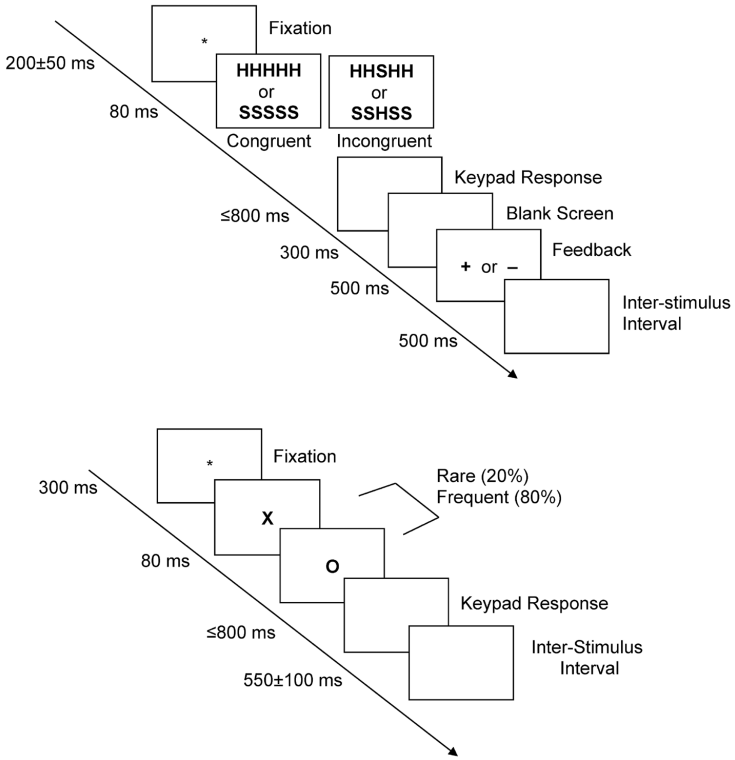

Figure 1.

Schematic of the stimulus display during the Eriksen Flanker Task (A) and Go/No-go Continuous Performance Task (B). For the Flanker Task, each trial began with a fixation asterisk followed by a congruent (50%) or incongruent (50%) letter string. Participants were allotted 800 ms for keypress response using dominant hand, after which feedback was presented. For the Go/No-go task, each trial began with a fixation asterisk followed by a letter stimulus. Participants were allotted 800 ms to respond. No feedback was provided between trials.

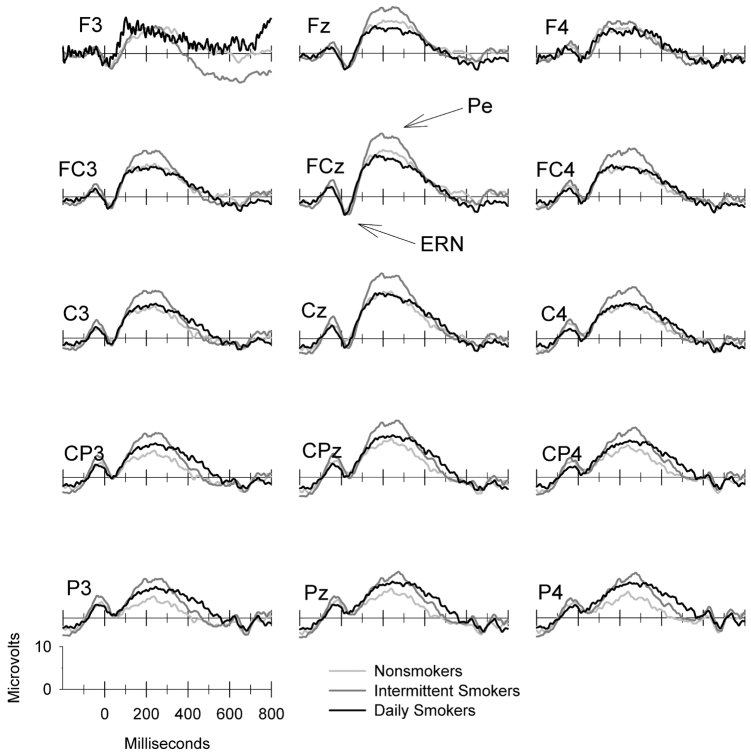

Figure 2.

Response-locked grand average ERP activity during error trials of the Eriksen Flanker Task for nonsmokers (light gray line), intermittent smokers (dark gray line), and daily smokers (black line).

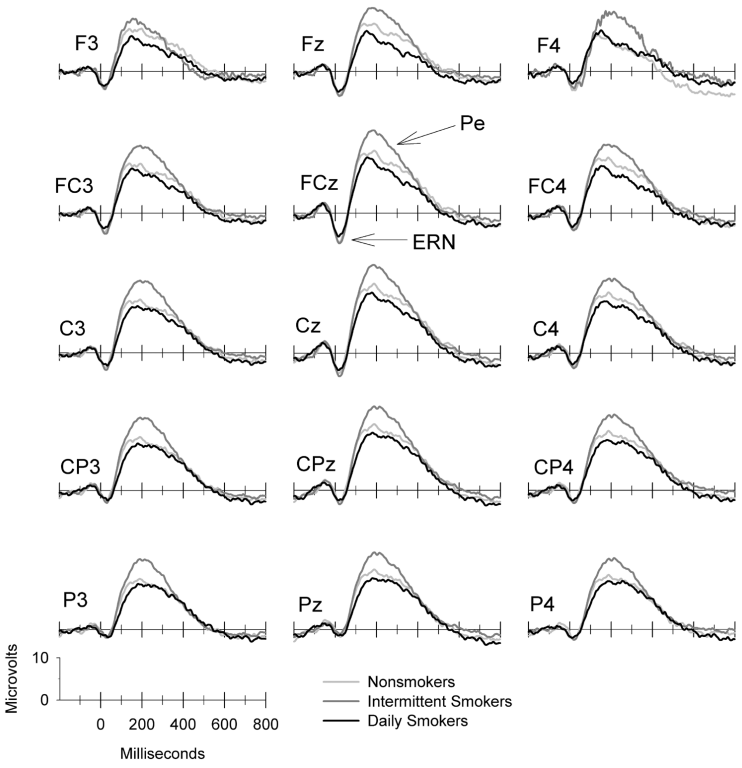

Figure 3.

Response-locked grand average ERP activity during error trials of the Go/No-go Continuous Performance Task for nonsmokers (light gray line), intermittent smokers (dark gray line), and daily smokers (black line).

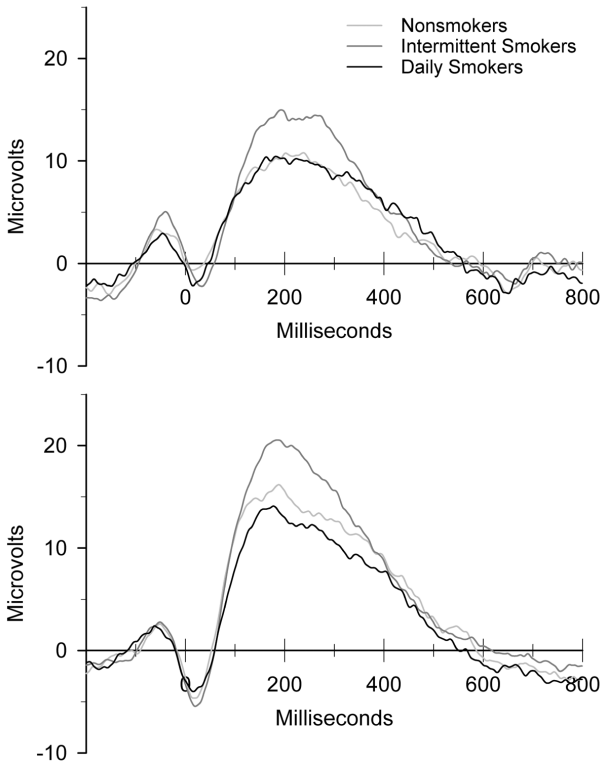

Figure 4.

Response-locked grand averages of ERP waveforms during error trials of the Eriksen Flanker Task (top) and Go/No-go Continuous Performance Task (bottom) for nonsmokers (light gray line), intermittent smokers (dark gray line), and daily smokers (black line) at the Cz electrode site.

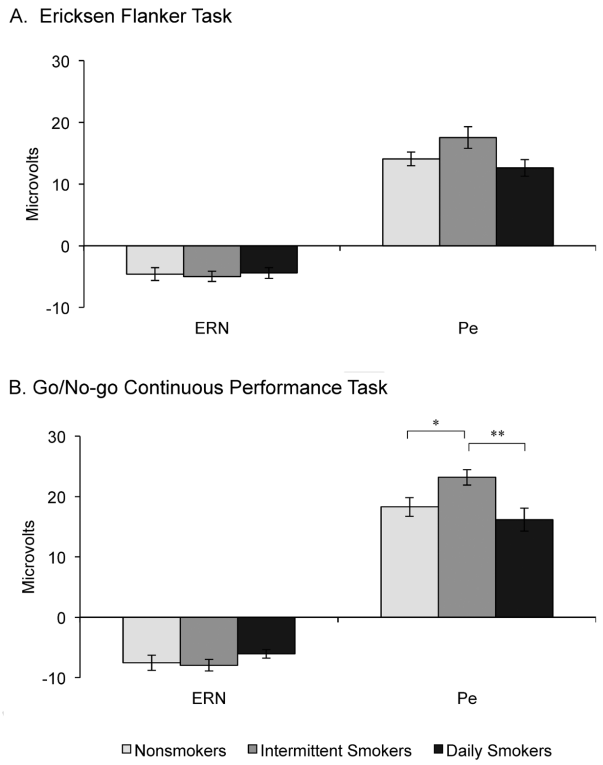

Figure 5.

Peak to peak measures of the ERN and Pe ERP components at the Cz electrode site during incorrect trials of the Eriksen Flanker (top) and Go/No-go Continuous Performance (bottom) tasks for nonsmokers (light gray), intermittent smokers (dark gray), and daily smokers (black). Error bars represent ±1 standard error. The following symbols represent significance: *$p < .05$, **$p < .01$.

Table 1

Demographics and Smoking Behavior.

	Nonsmokers (*n* = 30)	Intermittent Smokers (*n* = 31)	Daily Smokers (*n* = 22)	Analysis	*p*
Sex Male n (%)	14 (47%)	12 (39%)	13 (59%)	$X^2_{(2)} = 2.15$	.342
Age *M (SD)*	25.2 (4.3)	23.9 (4.4)	27.2 (5.3)	$F_{(2,80)} = 2.54$	.085
Education (years)	16.6 (2.0)[a]	15.8 (1.6)	14.8 (1.6)[b]	$F_{(2,80)} = 6.13$	.003
Caffeine use any *n* (%)	24 (80%)	28 (90%)	21 (95%)		
Drinks / week *M (SD)*	6.4 (6.4)	9.1 (6.3)	11.0 (6.5)	$F_{(2,80)} = 2.90$	.062
Alcohol use any *n* (%)	22 (73%)	30 (97%)	22 (100%)		
Drinks / night *M (SD)*	1.9 (1.0)[a]	3.1 (1.5)[b]	3.7 (2.0)[b]	$F_{(2,71)} = 8.28$	.001
Nights / week *M (SD)*	1.6 (1.0)	1.9 (1.0)	1.8 (0.9)	$F_{(2,71)} = .93$	.401
Any past drug use *n* (%)	5 (17%)	17 (55%)	11 (50%)	$X^2_{(2)} = 10.59$	.005
Marijuana	5 (100%)	17 (100%)	11 (100%)		
Other	0 (0%)	2 (12%)	4 (36%)		
Anxiety/Depression history *n* (%)	2 (7%)	8 (26%)	4 (18%)	$X^2_{(2)} = 4.02$	.134
Ethnicity *n* (%)					
Caucasian	20 (67%)	24 (77%)	18 (82%)		
Asian	8 (27%)	5 (16%)	3 (14%)		
Black	2 (7%)	1 (3%)	1 (5%)		
Biracial	0 (0%)	1 (3%)	0 (0%)		
CO ppm *M (SD)*					
Post	2.0 (0.8)[a]	2.8 (1.9)[a]	14.9 (8.8)[b]	$F_{(2,80)} = 57.49$	<.001
Pre	2.3 (1.4)[a]	3.7 (2.9)[a]	16.8 (10.8)[b]	$F_{(2,80)} = 45.66$	<.001
Change score	−0.3 (0.8)[a]	−0.9 (1.3)	−1.9 (3.9)[b]	$F_{(2,80)} = 3.36$	.040
WSWS Craving *M (SD)*					
Pre	1.0 (2.4)[a]	7.0 (6.5)[b]	11.5 (7.5)[c]	$F_{(2,80)} = 22.20$	<.001
Post	0.8 (2.0)[a]	6.3 (6.1)[b]	15.0 (7.3)[c]	$F_{(2,80)} = 43.56$	<.001
Change score	−0.2 (1.7)[a]	−0.7 (3.9)[a]	−3.5 (6.3)[b]	$F_{(2,80)} = 7.41$	.001
Age Initiation *M (SD)*		18.1 (1.7)[a]	16.3 (3.0)[b]	$F_{(1,51)} = 7.88$	.007
Years Smoked		5.6 (4.4)[a]	9.4 (6.4)[b]	$F_{(1,51)} = 6.69$	.013
NDSS Total Score		−2.6 (0.7)[a]	−0.1 (0.8)[b]	$F_{(1,51)} = 89.70$	<.001
FTND Total Score		0.5 (0.9)[a]	5.4 (1.3)[b]	$F_{(1,51)} = 272.52$	<.001
1. Cigarettes/Day		3.6 (1.6)[a]	16.1 (6.1)[b]	$F_{(1,51)} = 119.07$	<.001
2. Smoke within 5 min		0%	32%		
Smoke 5–30 min		3%	64%		
Smoke > 30 min		97%	5%		
3. Hate to give up first cig		13%	68%		
Hate to give up other cig		87%	32%		
Attempted to quit smoking		48%	95%		

Note. Means with different superscripted letters are significantly different, $p < .05$. Quantity of caffeine per caffeinated drink was not measured. Alcohol use data from one ITS is missing. Caffeine and alcohol use analysis included only those who used the substances. Drug use history refers to reported past use, which does not qualify as abuse or dependence under DSM-IV. Anxiety/Depression history refers to self-reported past episodes of anxiety or major depression. WSWS = Wisconsin Smoking Withdrawal Scale revised; FTND = Fagerstrom Test for Nicotine Dependence; NDSS = Nicotine Dependence Syndrome Scale. Cigarettes per day ranged from 2 to 10 for ITS and from 5 to 25 for Daily Smokers.

Table 2

Analysis of ERP measures for the behavioral control tasks.

	Nonsmokers	Intermittent Smokers	Daily Smokers	Analysis	*p*	*Partial* η^2
Eriksen Flanker Task						
# Error Trials	35 (20)	28 (17)	28 (14)	$F_{(2,61)} = 1.440$	.244	-
ERN Latency	21.4 (25.0)	33.0 (19.6)[a]	13.9 (25.6)[b]	$F_{(2,67)} = 3.949$	.024	.105
ERN Voltage	−4.6 (5.1)	−5.0 (4.1)	−4.4 (4.4)	$F_{(2,67)} = 0.100$	.905	-
Pe Latency	195.1 (48.4)	188.5 (42.8)	183.1 (42.5)	$F_{(2,67)} = 0.406$	.668	-
Pe Voltage	14.1 (5.5)	17.5 (8.7)	12.6 (6.1)	$F_{(2,67)} = 3.082$	.052	.084
Go/No-go Continuous Performance Task						
# Error Trials	30 (16)	30 (14)	30 (10)	$F_{(2,72)} = 0.002$	.998	-
ERN Latency	12.7 (21.9)	19.2 (15.9)	19.1 (16.1)	$F_{(2,72)} = 1.067$	.349	-
ERN Voltage	−7.5 (6.3)	−7.9 (5.2)	−6.1 (3.3)	$F_{(2,72)} = 0.836$	.438	-
Pe Latency	186.3 (46.7)	184.8 (40.8)	179.9 (44.0)	$F_{(2,72)} = 0.132$	.876	-
Pe Voltage	18.3 (8.0)	23.2 (6.8)[a]	16.2 (8.2)[b]	$F_{(2,72)} = 5.416$	.006	.131

Note. ERN analyses used the FCz electrode site and Pe analyses used the Cz electrode site. Values represent *M (SD)*. Means with different superscripted letters are significantly different, $p < .05$.

Table 3

Performance on inhibitory control measures.

	Nonsmokers	Intermittent Smokers	Daily Smokers
Eriksen Flanker Task			
Congruent RT	352.6 (59.1)	342.4 (38.1)	361.7 (51.6)
Incongruent RT	384.7 (69.7)	372.7 (38.3)	391.5 (54.0)
# Congruent errors	10.0 (8.5)	6.3 (5.9)	7.9 (4.8)
#Incongruent errors	22.9 (15.1)	19.8 (14.0)	20.8 (9.8)
Post-error slowing (ms)	50.8 (32.9)	63.6 (43.9)	65.4 (45.9)
Go/No-go Continuous Performance Task			
Frequent RT	239.3 (26.6)	230.1 (39.3)	250.2 (43.0)
Rare RT	286.9 (56.7)	275.1 (51.7)	291.5 (46.4)
# Frequent errors	5.2 (4.8)	4.9 (3.9)	6.4 (5.4)
# Rare errors	24.1 (14.7)	25.2 (12.4)	24.5 (8.3)
Post-error slowing (ms)	37.9 (34.9)	29.9 (25.1)	36.0 (36.3)

Note. Values Represent *M (SD)*. Groups did not differ on behavioral performance. RT = Response Time.